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EXAMINER	
WILSON, MICHAEL C	

ART UNIT	PAPER NUMBER
1632	

DATE MAILED: 07/06/2005

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	09/863,606	LISZIEWICZ ET AL.	
	Examiner	Art Unit	
	Michael C. Wilson	1632	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *29 May 2005*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *28-32* is/are pending in the application.

4a) Of the above claim(s) *32* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *28-31* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: _____.

DETAILED ACTION

Applicant's arguments filed 5-9-05 have been fully considered but they are not persuasive.

The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.

Claims 1-27 are cancelled. Claims 28-32 have been added. Claims 15, 16, 21-24, 26 and 27 are pending.

Claim 28 is substantially the same as claim 22 and should have been presented as claim 22 with the additions and deletions marked with underlining and brackets. This format is used to more quickly identify changes made to the claims.

Election/Restrictions

Claims 28-32 comprise inventions nonelected with traverse in the paper filed 5-12-03. A complete reply to the final rejection must include cancellation of nonelected claims or other appropriate action (37 CFR 1.144) See MPEP § 821.01.

The original restriction/election was limited to administering the patentably distinct combination of ddI (an RT inhibitor) and indinavir (a protease inhibitor). The restriction is being maintained because i) RT inhibitors, protease inhibitors and hydroxyurea have different structures and functions, ii) the species of RT inhibitors in claims 29 and 31 have different structures and inhibit RT using different mechanisms, iii) the species of protease inhibitors in claims 30 and 31 have different structures and inhibit protease using different mechanisms, and ii) the burden required to search

administering all combinations of RT inhibitor species and protease inhibitor species together with administering DNA encoding an immunogenic retroviral protein would be undue. Applicants' request for searches of other antiviral drugs is, therefore, denied.

Claim 32 is withdrawn from further consideration pursuant to 37 CFR 1.142(b), as being drawn to a nonelected invention, there being no allowable generic or linking claim.

Claims 28-31 are under consideration in the instant application only as they relate to administering antiretroviral drug therapy comprising ddI (an RT inhibitor) and indinavir (a protease inhibitor) until viral replication is suppressed, and then administering DNA encoding an immunogenic retroviral protein operably linked with a promoter.

Specification

The amendment to the paragraph bridging pg 22-23 and pg 23-24 has not been entered. The amendment is not based on the previous version of the paragraph. Applicants must make changes to the version filed 3-11-04 with the proper markings.

The amendment filed 3-11-04 remains objected to under 35 U.S.C. 132 because it introduces new matter into the disclosure. 35 U.S.C. 132 states that no amendment shall introduce new matter into the disclosure of the invention. The added material that is not supported by the original disclosure is as follows: the specification does not support the changes made to the paragraph bridging pg 22-23 or the paragraph

bridging pg 23-24. Applicant is required to cancel the new matter in the reply to this Office Action.

Claim Rejections - 35 USC § 112 - enablement

New Matter

Claims 28-31 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

The phrase "at least one immunogenic retroviral protein" in claim 28 is new matter. Support cannot be found in claim 1 as asserted by applicants.

The limitation of "BMS 23632" in claims 30 and 31 was not contemplated in the specification as originally filed. It is not readily apparent that BMS 23632 was available at the time of filing.

Enablement

Claims 28-31 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention for reasons of record.

Administering an antiretroviral drug therapy comprising ddI and Indinavir until retroviral replication is effectively suppressed is considered enabled because Finzi taught administering a reverse transcriptase inhibitor and a protease inhibitor suppressed retroviral replication (Finzi et al. Science. Nov. 14, 1997, Vol. 278, pg 1295-1300).

Claims 28-31 require administering DNA encoding an immunogenic retroviral protein after administering the antiretroviral drug therapy. The sole disclosed purpose for administering DNA encoding an immunogenic retroviral protein is to induce an immune response against the retroviral protein that is therapeutic (pg 2, lines 14-19). Therefore, the step of administering DNA encoding an immunogenic retroviral protein must be fully enabled for using the DNA to obtain a therapeutic immune response against the "immunogenic retroviral protein". However, the specification does not enable using DNA encoding an immunogenic retroviral protein to induce a therapeutic immune response against a retrovirus in a host.

Claims 28-31 are not enabled because the structure of the DNA encoding an immunogenic retroviral protein that provides a therapeutic immune response against the retroviral protein is not enabled.

The state of the art at the time of filing was that the combination of vector, promoter, route of administration, level of expression and target tissue required to obtain a therapeutic or prophylactic effect using gene therapy was unpredictable. Miller of record (1995, FASEB J., Vol. 9, pages 190-199) review the types of vectors available for *in vivo* gene therapy, and conclude that "for the long-term success as well as the

widespread applicability of human gene therapy, there will have to be advances...targeting strategies outlined in this review, which are currently only at the experimental level, will have to be translated into components of safe and highly efficient delivery systems" (pg 198, col. 1). Deonarain of record (1998, Expert Opin. Ther. Pat., Vol. 8, pg 53-69) indicate that one of the biggest problems hampering successful gene therapy is the "ability to target a gene to a significant population of cells and express it at adequate levels for a long enough period of time" (pg 53, 1st ¶). Deonarain reviews new techniques under experimentation in the art that show promise but states that such techniques are even less efficient than viral gene delivery (see page 65, first paragraph under Conclusion section). Verma of record (Sept. 1997, Nature, Vol. 389, pages 239-242) reviews vectors known in the art for use in gene therapy and discusses problems associated with each type of vector. The teachings of Verma indicate a resolution to vector targeting has not been achieved in the art (see entire article). Verma also teaches appropriate regulatory elements may improve expression, but it is unpredictable what tissues such regulatory elements target (page 240, sentence bridging columns 2 and 3). Crystal of record (1995, Science, Vol. 270, page 404-410) also reviews various vectors known in the art and indicates, "among the design hurdles for all vectors are the need to increase the efficiency of gene transfer, to increase target specificity and to enable the transferred gene to be regulated" (page 409).

The state of the art regarding treating retroviral infection was unpredictable. Stricker of record (Medical Hypotheses, June 1997, Vol. 48, pages 527-9) teaches that

attempts to develop a vaccine against HIV have been unsuccessful because HIV vaccines do not neutralize HIV (pg 527, last paragraph through all of pg 528). Overall, a lack of understanding about protective immunity to HIV in humans, the sequence variability of HIV and the rapid replication of HIV contribute the ineffectiveness of vaccines against HIV (Bangham of record, Nov. 29, 1997, Lancet, Vol. 350, pages 1617-1621; page 1617, top of col. 1).

The specification teaches a complex comprising i) manosylated PEI and ii) DNA encoding an immunogenic HIV protein operably linked to a promoter. Administration of the complex to a host after drug therapy was followed by an increase in CD4 cells then a decrease in CD4 cells (pg 53).

The specification does not provide adequate guidance for one of skill to use DNA encoding an "immunogenic retroviral protein" to induce an immune response capable of treating a retroviral infection. The results described in the specification are not considered therapeutic because the overall result does not result in a net increase in CD4 cells. In addition, it cannot be concluded that the DNA encoding a retroviral protein caused the initial increase in CD4 cells because the experiment did not include controls - animals that did not receive drug therapy or the gene complex. The specification does not provide adequate guidance indicating the increase in CD4 was caused by an immune response to the retroviral protein encoded by the DNA - the drug therapy could have caused the increase in CD4. The specification did not teach treating animals that were already infected or challenging the animals after they were given DermaVir. For administration of DNA encoding a retroviral protein to induce a therapeutic immune

response, the specification must overcome the unpredictability in the art by adequately describing the structure of the DNA used, the dosage and route of administration that results in a therapeutic effect or "immunization." Without such guidance it would require one of skill in the art undue experimentation to overcome the unpredictability in the art regarding gene therapy and retroviral therapy to determine the combination of elements required to obtain a therapeutic or prophylactic effect against retroviral infection using DNA. Therefore, the specification does not enable treating retroviral infection using DNA as claimed.

The specification states:

> "The comparison of the rate of viral load rebound among those animals undergoing STI-HAART early after infection (Lori, F. et al. Control of SIV rebound through structured treatment interruptions during early infection. Science 290, 1591-1593. (2000)), those initiating STI-HAART during AIDS, and the same animals treated with STI-HAART plus DermaVir$_{SHIV}$ revealed an interesting pattern. The rate of viral rebound during consecutive HAART interruptions, that was unchanged before the initiation of vaccine therapy, decreased sharply after vaccination, and became remarkably similar to that observed in the animals treated with STI-HAART early after infection (Fig. 14). These results suggest that DermaVir$_{SHIV}$ therapy can improve the control of virus replication during interruption of HAAR." (pg 53, lines 18-27).

HAART therapy as described in Lori of record (2000) is PMPA (tenofovir, an RT inhibitor), ddI (didanosine, an RT inhibitor) and hydroxyurea (pg 1591, col. 3, lines 10-18). STI-HAART is structured treatment interruptions of HAART therapy.

The examiner agrees that the interrupted administration of PMPA, ddI and hydroxyurea followed by administration of DermaVir$_{SHIV}$ (AIDS(DermaVir)) in Fig. 14 shows decreased viral rebound as compared to interrupted administration of PMPA, ddI and hydroxyurea (AIDS(HAART)).

The claims are being considered as they relate to administering ddI and indinavir followed by a gene complex; however, the example is limited to administering PMPA, ddI and hydroxyurea followed by a gene complex. The combination of administering drugs plus DermaVir$_{SHIV}$ in the example does not correlate to administering ddI and indinavir plus DermaVir$_{SHIV}$. The specific combination of DermaVir$_{SHIV}$ with PMPA or hydroxyurea may have decreased viral rebound in the example. Perhaps the combination of two different RT inhibitors in the example with DermaVir$_{SHIV}$ decreased viral rebound (PMPA and ddI have different structures and different mechanisms of action (see DeClercq, Current Medicinal Chemistry, 2001, Vol. 8, pg 1543-1572; ¶ bridging pg 1553-1554; nucleotide vs. nucleoside analogues; "PMPA only needs two phosphorylation steps to be converted to the active metabolite"). The example does not use indinavir or any other protease inhibitor. The decreased viral rebound effect in the example may be a synergistic effect obtained only in the presence of PMPA, PMPA and ddI, or hydroxyurea. Therefore, one of skill would not expect DermaVir$_{SHIV}$ to decrease viral rebound after administering ddI and indinavir based on the example, which is limited to administering ddI, PMPA and hydroxyurea followed by DermaVir$_{SHIV}$.

Furthermore, the claims encompass administering continuous HAART followed by DermaVir$_{SHIV}$; however, the example is limited to interrupted HAART. The specification does not correlate decreasing viral rebound obtained by interrupting HAART followed by DermaVir$_{SHIV}$ with expected results obtained by administering continuous HAART plus DermaVir$_{SHIV}$ (i.e. the virus does not rebound during continuous

HAART). Therefore, the mode of drug delivery in the example does not correlate to any mode of delivery as broadly encompassed by claim 21.

The claims encompass delivering any gene complex comprising DNA encoding any immunogenic retroviral protein; however, the example is limited to DermaVir$_{SHIV}$. The specification states:

> "DermaVir$_{SHIV}$ is a glucose-water solution containing a plasmid DNA as an active ingredient and polyethylenimine-mannose (PEIm) as an adjuvant (See Example 12). One therapeutic application contained 0.1 mg DNA capable of expressing all but the integrase protein of the Simian-Human Immunodeficiency Virus (SHIV). DermaVir$_{SHIV}$ was formulated to transduce Langerhans cells located in the epidermis and it was applied on the surface of the skin of the animals. We have shown that these Langerhans cells are triggered to migrate to the lymph nodes, mature to dendritic cells and present SHIV antigens to naïve T cells. After SHIV-specific activation of naïve T cells in the lymph nodes, DermaVir$_{SHIV}$ initiated potent SIV-specific T cell-mediated immune responses in uninfected monkeys (See Example 12)" (pg 52, lines 1-9).

The specification does not correlate the results obtained with DermaVir$_{SHIV}$, which expresses all retroviral proteins except integrase, to any DNA encoding any immunogenic retroviral protein as broadly claimed, specifically DNA encoding one immunogenic retroviral proteins, such as gp120. The expression of all retroviral proteins may be essential to induce the proper immune response and decrease viral rebound. (see pg 52, lines 1-9).

Not only is the gene complex itself much narrower in scope than the gene delivery complex claimed, the mode of delivery described in the specification is limited to dermal administration.

In conclusion, the example on pg 53 is much narrower than claim 21 in the types of drugs administered, the mode of delivery of the drugs, the gene complex being

delivered and the mode of delivery of the gene complex. Decreasing viral rebound after interrupting two RT inhibitors and hydroxyurea cannot even be extrapolated to administration of ddI and indinavir because the combination of drugs in the example may have allowed DermaVir$_{SHIV}$ to function.

Applicants argue, "the examiner appears to be operating under a misunderstanding of the applicable law. The disclosure in a patent application is required to enable one skilled in the art to make or use the claimed invention. There is no requirement for an inventor to conform to somebody else's idea of what might be required. Further, all the disclosure of the application must be relied upon for enablement of the invention and not just the specific examples used to illustrate the invention. The Claims are not limited merely to the specific materials used in the Examples. In addition, the Examiner appears to be operating under a misunderstanding of fact: a therapeutic immune response is unquestionably demonstrated in the text."

Applicants' argument is not substantial. The examiners rejection reviews the teachings of the specification in context of the Examples and the teachings in the art at the time of filing and correlated them with the Wands factors of enablement. Applicants have not pointed to one error in the examiner's logic or in the analysis of the law or the teachings in the specification.

Applicants argue Miller, Deonarain, Verma, Crystal, etc. "were wrong, and that is why they are not the inventors of the present case." Applicants' argument is not

substantial and is illogical. Applicants' logic that Miller, Deonarain, etc. were not the inventors of the present case because they "were wrong" is flawed, and the reasons why they were not inventors are irrelevant. Applicants' have not provided to any evidence that those numerous skilled artisans at the time of filing were wrong.

Applicants arguments in the last 14 lines of pg 9 of the response are not persuasive because they discuss fluctuations in CD4 cell numbers without discussing how the DNA contributed to a therapeutic immune response or specifically to CD4 cell numbers. The basis of the rejection is that the specification does not provide adequate guidance indicating the DNA contributed to any therapeutic effect.

Applicants' argument in the first paragraph of pg 10 regarding the animals being infected already is moot. Applicants do not discuss how the specification enables one of skill to use the DNA to induce a therapeutic immune response – the basis of the rejection.

Applicants argue controls died long before this set of experiments were begun (pg 10, 2nd ¶). Applicants' argument is moot. "This set of experiments" (i.e. Example 13) needed their own set of controls. Specifically, a group that was capable of determining whether the DNA contributed to the increased CD4 counts. Without evidence to the contrary, given the lack of DNA vaccines capable of increasing CD4 counts, the DNA administered by applicants did not contribute to the increased CD4 counts. Applicants fail to describe any logic as to how one of skill could conclude without the proper control that the DNA contributed to the increased CD4 and was not solely caused by the anti-retroviral therapy.

Applicants' argument in the paragraph bridging pg 10-11 discusses the examiners position regarding the lack of correlation between the antiretroviral drugs in Example 13 and ddI and indinavir currently under consideration in the claims. Applicants' state: "[t]his possibility has been eliminated by the progression of the experiment." "This possibility" appears to refer to the examiner's position; however, the meaning of applicants' statement is unclear. It cannot be determined what has been "eliminated by the progression of the experiment." PMPA or hydroxyurea used in the Example 13 do not correlate to ddI and indinavir as currently under consideration because they have different structures and different modes of action, and because they may act synergistically with the DNA while ddI and indinavir may not.

The scope of the gene complex in parent application 6,420,176 is moot because the claims in '176 are directed toward the gene complex, not a method of treating a retroviral infection using the gene complex. The gene complex of '176 can be used to transfect cell in vitro and is not limited to being used to treat a retroviral infection as now specifically claimed.

The prior art

Claims 28-31 are free of the prior art as they relate to administering antiretroviral drug therapy comprising ddI (an RT inhibitor) and indinavir (a protease inhibitor) until viral replication is suppressed, and then administering a DNA complex comprising a) DNA encoding an immunogenic retroviral protein operably linked with a promoter; and b) mannosylated polyethylenimine. The prior art did not teach or suggest administering

ddI and Indinavir until viral replication is effectively suppressed, and then administering a gene delivery complex as claimed. Finzi (Science. Nov. 14, 1997, Vol. 278, pg 1295-1300) taught administering reverse transcriptase inhibitors and protease inhibitors to HIV patients. However, Finzi did not relate to administering DNA encoding the marker protein luciferase to the brain of mice as taught by Boussif (PNAS, Aug. 1995, Vol. 92. pg 7292-7301) of record, administering DNA encoding a marker protein to cells *in vitro* as taught by Zanta (Bioconjugate Chem. 1997. Vol. 8. pg 839-844) of record, administering DNA encoding a marker protein to cells *in vitro* as taught by Behr (US Patent 6,013,240) of record, or administering virus encoding integrase-defective HIV to cells *in vitro* as taught by Cara (Virology, 1995, Vol. 208, pg 242-248).

The claims have only been searched for ddI and indinavir in combination with the gene complex.

Double Patenting

Claims 28-31 are rejected under the judicially created doctrine of obviousness-type double patenting as being unpatentable over claims 1-14 of U.S. Patent No. 6,420,176 in view of the disclosure of 6,420,176 for reasons of record. The claims of '176 are directed toward a gene delivery complex comprising DNA encoding an immunogenic protein operably linked to a promoter and monosylated polyethylenimine. The claims of '176 do not require administration as required in the instant claims or administration of antiretroviral drug therapy. MPEP 804 states the specification may be used as a dictionary to learn the meaning of a term in the patent

claim. In this case, one of skill would look to the specification to determine the asserted utility of the product. The disclosure taught administering the gene delivery complex after suppressing viral replication using antiretroviral drug therapy (col. 12, lines 11-51, see especially lines 20-27). Thus, it would have been obvious to one of ordinary skill in the art at the time the invention was made to administer the gene delivery complex in combination with drug therapy as claimed.

Applicants discuss what was desired in a vaccine but does not discuss why the parent application fails to teach one of skill to use the DNA to treat retroviral infection. The parent application states the DNA can be used to treat retroviral infection. Col. 12, line 19, does not indicate applicants "limitations of the use of the vaccine" because it states the DNA "can strengthen the immune [system]."

Applicants' discussion of the examiners response to previous arguments regarding "unexpected results" is moot (last paragraph on pg 14 of response). Applicants have not elaborated on the previous "unexpected results" arguments. The examiner was merely addressing applicants' misplaced "unexpected results" argument to the best of his ability.

Claims 28-31 are provisionally rejected under the judicially created doctrine of obviousness-type double patenting as being unpatentable over the claims of copending Application No. 10/081922 for reasons of record. Although the conflicting claims are not identical, they are not patentably distinct from each other because they overlap in scope. Applicants have not addressed this rejection.

Conclusion

Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

No claim is allowed.

Inquiry concerning this communication or earlier communications from the examiner should be directed to Michael C. Wilson who can normally be reached at the office on Monday, Tuesday, Thursday and Friday from 9:30 am to 6:00 pm at 571-272-0738.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to (571) 272-0547.

Patent applicants with problems or questions regarding electronic images that can be viewed in the Patent Application Information Retrieval system (PAIR) can now contact the USPTO's Patent Electronic Business Center (Patent EBC) for assistance. Representatives are available to answer your questions daily from 6 am to midnight (EST). The toll free number is (866) 217-9197. When calling please have your application serial or patent number, the type of document you are having an image problem with, the number of pages and the specific nature of the problem. The Patent

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If attempts to reach the examiner are unsuccessful, the examiner's supervisor, Ram Shukla, can be reached on 571-272-0735.

The official fax number for this Group is (571) 273-8300.

Michael C. Wilson

MICHAEL WILSON
PRIMARY EXAMINER